

Public Health Then and Now

Public Health Nihilism vs Pragmatism: History, Politics, and the Control of Tuberculosis

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ABSTRACT

Tuberculosis (TB) began to decline in the Western world in the mid- to late 1800s. In the United States, the disease receded until the mid-1980s, when that trend was reversed. Although the TB epidemic in the United States subsided in response to public health interventions, it sparked a controversy regarding the relative value of targeted public health measures vs broad social reform. That controversy, which echoed earlier debates calling for structural reform over public health programs, was further strengthened by the historical and demographic studies of Thomas McKeown. His influential thesis maintains that clinical and primary prevention efforts had little effect on TB mortality.

In this paper, the historical literature is used to examine whether public health had a significant impact on the decline of TB mortality rates in several countries. Specifically, the paper describes the arguments for and data affirming the efficacy of 2 major public health interventions over time: segregation of those infected with pulmonary TB and eradication of bovine TB. This review finds support for the hypothesis that public health measures, along with other factors, led to falling rates of TB mortality beginning in the late 19th century. (*Am J Public Health*. 1998;88:1105–1117)

Tuberculosis (TB)—the 17th century's "captain of all these men of death" and the 19th century's "white plague"—began to decline in many countries during the mid- to late 1800s. In the United States, the disease steadily receded until the mid-1980s, when that trend was reversed. From 1985 through 1991, the number of reported TB cases in the United States increased 18%,¹ provoking governmental agencies to consider such targeted public health interventions as isolation of infected persons and directly observed therapy. Although directly observed therapy had been used successfully in Madras and Hong Kong decades before the TB epidemic in the United States, public health officials here remained skeptical of the measure until the 1990s.² However, with the surge in TB cases, the rise in multiple-drug-resistant forms of the disease, and the close association of the disease with the AIDS epidemic, the political and cultural climate of TB control underwent a radical shift. Congress, reacting to public fear that untreatable TB would spread into the general population, greatly increased funding for control measures.³ Suddenly, directly observed therapy became the centerpiece of public health efforts to stem the disease.⁴

Although the TB epidemic in the United States subsided in response to directly observed therapy and complementary public health interventions,⁵ it sparked a controversy regarding the relative value of targeted public health measures vs broad social reform.⁶ Some saw the deterioration of the public health infrastructure since the 1960s—the loss of public financing for TB screening, follow-up, and treatment—as the predisposing cause of the reemergence of TB in the 1980s.⁷ The epidemic was subsequently the tragic result of HIV disease and insufficient public planning and investment.⁸ Adherents to this view argued that "a structured commitment to find an adequate public health infrastructure could control

tuberculosis" and, moreover, could do it "independent of the need for broader social change."⁹ Others, in contrast, charged that public health campaigns had failed to control TB effectively because the "control measures have dealt with individual health habits rather than addressing the underlying poverty that predisposes individuals to tuberculosis."¹⁰ Consequently, such limited interventions could, by themselves, achieve only a partial and temporary success.¹¹

Controversy between supporters of limited, targeted actions and those who urge more sweeping changes is not new, particularly in the case of TB; here current arguments echo and are reinforced by earlier debates calling for structural reform in terms of public health interventions.¹² Since the mid-1960s, that brief for broad reform has been further strengthened by the historical and demographic studies of Thomas McKeown, who maintained that incremental secular changes in the population's standard of living—especially better nutrition—accounted for the bulk of the decline in TB mortality since the mid-19th century. Public health efforts were nugatory against airborne disease. McKeown's influential thesis, which finds little use for clinical and public health initiatives, is partially reinforced by recent historical works that cast

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doubt on the efficacy of public health programs in combating TB.

The belief that broad social reforms (such as increases in real wages or the standard of living), rather than limited public health programs, profoundly reduce rates of disease has been described recently by Ronald Bayer as "public health nihilism."¹³ His coinage makes reference to the therapeutic nihilism of the early-19th-century New Vienna School, whose core idea was that physicians might diagnose and describe disease, but they could not cure it. Bayer argues that public health nihilism has been central to debates in such areas as TB, population and family planning,¹⁴ and mental health.¹⁵ His term for those who dismiss targeted interventions is evocative, resonating with historical doubt about the efficacy of limited interventions on disease in individuals and populations.

Are targeted public health approaches without historical support? Here we use the historical literature to examine whether such efforts may have had a significant effect on the historical decline in TB mortality rates in the United States, Britain, and France. Specifically, using the extensive historical studies of TB, we describe the arguments for and data supporting the efficacy of 2 major public health interventions over time: segregation of those infected with pulmonary TB and eradication of bovine TB. We find that this literature weakens the arguments of McKeown and those who share his perspective in favor of improved general well-being as the fundamental cause of the fall in TB mortality.

We further argue that, over time, historians framed the issue of the relationship between public health and TB mortality on the basis of their political sensibilities and values. These sensibilities and values determined the questions each generation raised about TB and the solutions they were prepared to entertain.

Our review also suggests the need for multifactorial models, based on local studies, to explain that historical decline. Such models must include factors other than public health measures, including data on nutrition, occupational health, and housing. They should also differentiate between historical periods. Targeted interventions, especially patient isolation, probably became more efficacious as TB endemicity declined and the disease became more localized, a problem of smaller social groups and families. In addition, the types of public health campaigns mounted in the pre-bacteriological and bacteriological eras were different. Ultimately, a broad series of studies is required to explain the decline of TB. There is always a danger to drawing policy recommendations regarding disease prevention from a single historical study, a limited period of time, or a single variable rather than

from a broad spectrum of studies considering the history of a complex disease in different times, places, and populations.

Background: Tuberculosis and the McKeown Thesis

Thomas McKeown made a signal contribution to demographic, epidemiologic, and historical studies by attempting to answer a challenging question: Why did mortality from infectious disease decline dramatically during the 19th and 20th centuries?¹⁶ The significance of McKeown's question and of the scholarship invested in his answer has had an impact on a generation of social scientists, historians, and policymakers.¹⁷ His conclusions were most recently recapitulated, for example, in a new history of the rise and decline of TB in Japan, whose author noted, "Nutrition is one of the most powerful of all socially and environmentally determined influences on the development or retardation of active tuberculosis. . . . [N]either medicine nor public health measures had a significant impact on mortality from the disease until after World War II."¹⁸

McKeown, in studying declining death rates in England and Wales, pinpointed TB and other airborne disorders. These contributed more, he asserted, than all other infectious diseases combined to the fall in crude mortality between 1848 and 1971.¹⁹ To explain this general reduction in mortality, McKeown laid out a list of mutually exclusive possible causes, starting with changes in the virulence of microorganisms, continuing on to scientifically based clinical interventions, and ending with an increase in the quality and level of nutritional intake. These causes (including the development of hospitalization, chemotherapy, and vaccination) he successively eliminated, until he was left with 2 hypotheses: public health (sanitation) reforms and a general improvement in nutrition. McKeown then reasoned that better sanitation could have affected only water- and foodborne diseases, which contributed a far smaller fraction to the crude mortality decline. By logical elimination, then, McKeown found that improved host resistance, as a consequence of better nutrition, best explained the decline in airborne disease, TB included, in the period under consideration.

McKeown lacked direct evidence to support this last hypothesis; instead, having eliminated other causes, he initially left the lion's share of mortality reduction, by default, to better nutrition.²⁰ In another work, he offered nutrition as a hypothesis that, based on circumstantial evidence, should be made to stand "the test of critical examination in a variety of circumstances over an extended period."²¹ To buttress his hypothesis, he provided limited

data derived from animal models, inferences concerning increased food production in England and Wales in the 19th century, and current general experience in developing countries.²²

McKeown's opus, although influential, has drawn sharp criticism. He has been taken to task for his data analysis and for his thesis that improvement in nutrition was the predominant cause of the decline in TB mortality in the 19th and 20th centuries. In both his early and later work, McKeown treated nutrition as "the most significant feature" of a rising standard of living during those centuries; his presumption was that amelioration in health status stemmed from a changing economy rather than a set of specific acts engineered by human agency. Critics have responded by asserting the role of such agency, particularly in the form of broad, socially based public health measures.

Simon Szreter, a British historian, finds that the reasons for the decline in infectious-disease-specific mortality are more complex than McKeown's hypothesis allows. He argues that once airborne diseases are studied individually, rather than in the aggregate as McKeown studied them, the disease-specific causes of mortality reduction vary; for example, the streptococci associated with scarlet fever, by McKeown's own admission, may have decreased substantially in virulence during the 19th century. Indeed, of all the airborne diseases, only TB could have been affected by population-level malnutrition.²³

By demonstrating that TB began to decline later in the century than McKeown allows, Szreter concludes that in the instance of TB, as with other diseases, there were multifactorial reasons for the downward trend in mortality. Szreter cites the existence of intercurrent infections and occupational hazards that weakened host resistance to TB and of overcrowding and poor ventilation in work and home environments that enhanced transmission of the disease.²⁴ He suggests that these factors were removed not only by rising real wages and better nutrition but by political and social action associated with the public health movement, "a complicated history of struggle and pressure for relevant clauses in Factories and Workshop Acts, Housing and Crowding Acts, and the enforcement of building regulations and by-laws."²⁵ This is the prebacteriological public health campaign that attacked the unhygienic environmental and behavioral determinants of health.

While Szreter bases his critique and alternate hypothesis on an analysis of McKeown's original data, local studies of TB mortality in Britain support the role of broad environmental and political measures in controlling TB.²⁶ Anne Hardy finds that housing policy, possibly nutrition, public health education, and pro-

portionate declines in workers employed in high-risk industries help account for the dropping rate of TB mortality in London in the 19th century.²⁷ In his study of Glasgow in the 20th century, Neil McFarlane contends that improved housing, rather than better nutrition, helps account for the decline in pulmonary consumption.²⁸

Studies of other locales also support the role of environmental measures in reducing infectious disease mortality, including that associated with TB. Philip Curtin's study of British and French data from military posts on the continent and in Algeria, the British West Indies, and southern India documents a "mortality revolution" during the 1850s. Curtin finds that a sanitary engineering campaign substantially reduced mortality within the military from a number of water-borne diseases, first overseas and then at home.²⁹ Significantly, by controlling for nutritional intake and social class (limiting the study of mortality to working-class men in the military), Curtin's work offers unique insight into the control of TB.

While TB was responsible for the majority of deaths among troops stationed in Britain and France, the rates of TB mortality among troops abroad were much lower.³⁰ Although Curtin maintains that the cause of the differential decline "remains obscure," he proposes that improved ventilation—in this instance, in the troops' barracks—may account for lower TB-specific mortality rates in overseas posts. He discounts nutrition as a reason for "the enormous differences between death rates [from TB] at home and those overseas,"³¹ since diet within the ranks did not vary by posting.

One historian studying the many causes of the decline of TB observes that in "the debate between McKeown and his critics, the jury is still out."³² Although McKeown's work suffers from distinct weaknesses, so does that of his critics. These critics, who stress the role of human agency in the form of public health interventions, lack a body of valid quantitative data to support their position. To explain declines in TB mortality rates—a quantitative outcome—some quantitative measurement and analysis of the hypothetical causes would strengthen the critics' arguments. Many propose multifactorial models to explain the progressive fall in TB mortality rates; in the absence of data, however, they cannot test for the separate effect of each of the independent variables—housing, ventilation, sanitation, working conditions, nutrition, or intercurrent infections—while controlling for the others. Finally, unlike McKeown, his critics tend to focus on the 19th century, the prebacteriological era in particular. Consequently, to counter McKeown's nutrition theory, they offer public health models specific to that era: environmental measures targeting mortality in gen-

eral rather than TB in particular. Only a few critics have posited public health models that incorporate the insights of bacteriology: that TB is a contagious disease that requires case finding and the disruption of transmission through patient education and isolation. We turn now to those writers who evaluate McKeown from the perspective of the bacteriological revolution.

Segregation of the Infected: The Sanatorium

First established in Germany (Silesia) in the mid-19th century, sanatoriums were meant to cure TB through the agency of fresh air and exercise, diet, and graduated labor. In the 20th century, sanatoriums incorporated clinical interventions such as tuberculin treatment and pneumothorax. Historians, particularly the British, have challenged the clinical efficacy of these institutions. Nancy Tomes, in her recent review of TB literature, posits that British scholars, "writing in McKeown's shadow," feel compelled "to assess the relative importance of medical versus non-medical factors in the decline in mortality."³³ In addition, historians challenge whether sanatoriums, by isolating tuberculous patients, were useful agents of primary prevention, as Robert Koch and other contemporary physicians argued they should be.³⁴ Their skepticism is rooted in chronology.³⁵ Mortality from TB began to decline in a continuous fashion in Great Britain sometime between the 1830s and the 1880s³⁶; in the United States, that decline started in the 1860s or 1870s.³⁷ Yet sanatoriums and other special institutions for the care and isolation of patients with TB did not proliferate until after Robert Koch's discovery of the mycobacterium in 1882.³⁸

In Britain, which adopted the sanatorium late in the 19th century, there was great confidence in the curability of TB by "open-air" methods.³⁹ With cure as a goal, sanatoriums and voluntary hospitals carefully selected early cases of TB over chronic "incurables." The state sanatoriums, founded after the National Insurance Act of 1911, applied the same rules of patient selection.⁴⁰ By the first decade of this century, the clinical success rate of the sanatoriums had been found to be far lower than expected, with more than half of treated patients dead 5 years after discharge.⁴¹ The consequence of this medical emphasis was, in the view of current British historians such as Linda Bryder and F. B. Smith, a sad waste. Writes Smith: "It was unfortunate that a coterie of medical practitioners captured the tuberculosis specialty in the late 1880s and thereafter set the terms by which the disease was comprehended. Public money could and should have been diverted to trying preven-

tive measures and to helping severely impoverished cases and their families."⁴²

Similarly, American historians have often held that sanatoriums, because of their scope and design, could not have successfully isolated infectious individuals. For example, Sheila Rothman, in describing the coercive, punitive system of treatment and isolation facilities created by Hermann Biggs in New York City shortly after the turn of the century, argues that institutional organization and philosophy were fundamentally flawed. And Otisville, the municipal sanatorium opened in 1906, was "spartan, austere, and sometimes punitive."⁴³ Patients, accordingly, typically left the institution shortly after arriving.⁴⁴ Those institutions created to hold recalcitrant, intractable infectious patients by force, such as the hospital for the tubercular poor on Blackwell's Island in the East River and Riverside Hospital, established on North Brother Island, were also failures. According to Rothman, lacking the medical supplies, the necessary staff, and the facilities to maintain discipline, such "institutions...were too prison-like to be hospitals and too hospital-like to be prisons."⁴⁵

Barbara Bates, another American historian, bases her conclusions about the inefficacy of the relatively extensive system of sanatoriums in Pennsylvania on the limited scope of institutional care before the 1920s. She concludes that "[i]nstitutionalization...was not the leading cause of Philadelphia's falling death rate. The decline of tuberculosis began before that form of segregation and continued fairly steadily thereafter until 1916."⁴⁶ According to Bates, the system of charity and religious hospitals and sanatoriums created before the turn of the century simply did not house a significant portion of the tubercular. Those who did receive institutional care usually chose to die at home, undermining the benefits of segregation.

As these sanatoriums and hospitals were supplemented and eventually supplanted by state institutions during the early 20th century,⁴⁷ care of the tubercular did increasingly move out of the home, especially after 1910.⁴⁸ Still, sanatoriums fell short on their promise to isolate the infected. Institutions under financial pressure, for example, discharged patients who seemed improved. Indeed, by 1914 Philadelphia's Free Hospital for Poor Consumptives (later the White Haven Sanatorium Association) was limiting the maximum stay for any individual to 14 weeks.⁴⁹ In addition, the diagnosis was usually made late in infection; once diagnosed, patients continued to wait long periods before they could enter a sanatorium owing to lack of space.⁵⁰

Despite her argument concerning its negligible public health effects, Bates' own data

allow a reconsideration of the benefits of isolation. Bates observes that the rate of declining mortality usually signals the efficacy of any particular intervention. Interestingly, Bates' data show that the rate of decline increased perceptibly between 1880 and 1900, the period in which the first sanatoriums opened.⁵¹ She also notes that the TB death rate fell somewhat more rapidly after 1920; in this instance, she believes that "institutionalization may have had an impact," although segregation "cannot be separated from other possible causes."⁵²

Ultimately, Bates' primary concern—like that of most American historians—is to document the patients' experience of TB, not McKeeown's thesis or even the efficacy of public health measures.⁵³ The public health effect of patient isolation is, however, central to population-level, quantitative histories of TB. Leonard Wilson, for example, resurrects and further supplements the statistical analyses of Sir Arthur Newsholme, the medical officer of health in Brighton, England, and president of the Epidemiological Section of the Royal Society of Medicine. In work published from 1903 through 1908, Newsholme, according to Wilson, found that the segregation of individuals suffering from advanced pulmonary TB (phthisis) was responsible for the decline in TB mortality in England and Wales, Scotland, Prussia, and New York City. Newsholme's analyses indicated that improvements in nutrition and standards of living (measured by the course of wheat prices, among other indices), although strongly correlated with decreasing deaths from consumption in Great Britain, were only moderately to poorly correlated with declining death rates in Prussia and France; moreover, they were inversely correlated with phthisis mortality in Ireland (see Figure 1). Like modern demographers, Newsholme observed that the pattern of declining TB mortality and rising living standards in England and Wales was not generalizable to other countries.⁵⁴ If there existed strong, universal determinants of the fall in TB mortality rates across nations, then one had to look elsewhere.

Newsholme's solution, according to Wilson, was based on his observation that a growing proportion of consumptives were segregated in British Poor Law infirmaries and workhouses. The poor, who were at highest risk of TB (and further impoverishment), were sent to those institutions rather than to the relatively new and scarce sanatoriums.⁵⁵ Newsholme reasoned that the segregation of consumptives with active disease, by diminishing the transmission of infection, must have had a significant effect on TB incidence and mortality rates.

A comparison of Ireland with England, Wales, and Scotland provided Newsholme with a "natural experiment" to test his hypoth-

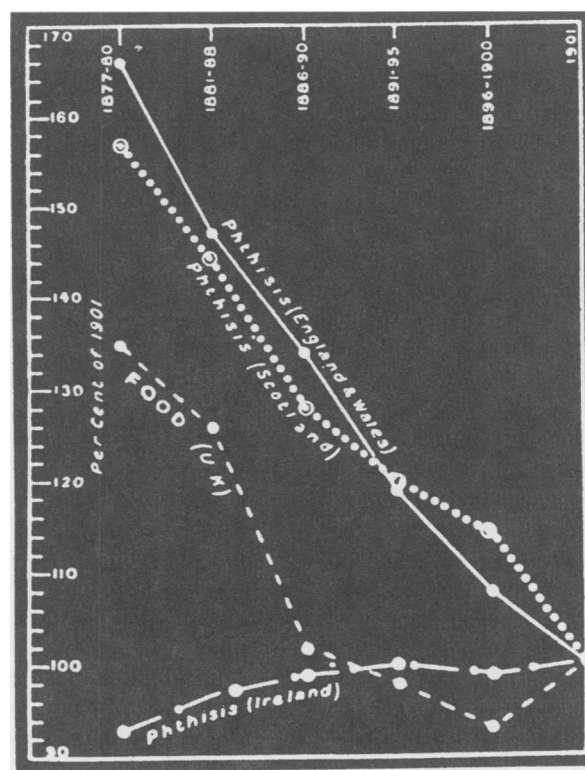


FIGURE 1—Newsholme's graph comparing phthisis mortality in England and Wales, Scotland, and Ireland with the cost of food in the United Kingdom (including Ireland) from 1877 to 1901. From Arthur Newsholme, "An Inquiry into the Principal Causes of the Reduction in the Death-Rate from Phthisis During the Last Forty Years, with Special Reference to the Segregation of Phthisical Patients in General Institutions," *Journal of Hygiene* 6 (July 1906): 336.

esis further. In Britain, both typhus—a contagious louse-borne disease—and phthisis mortality rates had declined since 1870. Although typhus rates had also decreased in Ireland, phthisis mortality rates continued to rise. Could this be due to varying social conditions? Newsholme, according to Wilson, found that Ireland, like England, Wales, and Scotland, enjoyed improved housing, growing real income, and lower food costs during the last 30 years of the century. Then why the paradoxical differences between Ireland and the rest?

Newsholme argued that the cause lay chiefly in the administration of the poor laws in Ireland. On one hand, the Irish laws sufficiently immobilized the population to prevent typhus-infected paupers from migrating in search of work and thereby spreading disease.⁵⁶ On the other hand, a far higher proportion of the Irish poor received "outdoor relief" (relief in the home) rather than "indoor" or institutional benefits. Consequently, most impoverished Irish consumptives depended on domestic treatment rather than segregated

confinement as in Britain, thereby considerably enhancing the transmission of phthisis within families. Newsholme also found this pattern in Norway, a nation in which only a small proportion of the sick were institutionally treated.

Wilson maintains that, by demonstrating the constancy of the relationship between declining TB mortality and the increased segregation of infected patients and by documenting the inconsistent association between TB mortality and indices of improved social conditions across a number of countries, Newsholme successfully eliminated population living standards as a key determinant in understanding the decline in TB mortality.

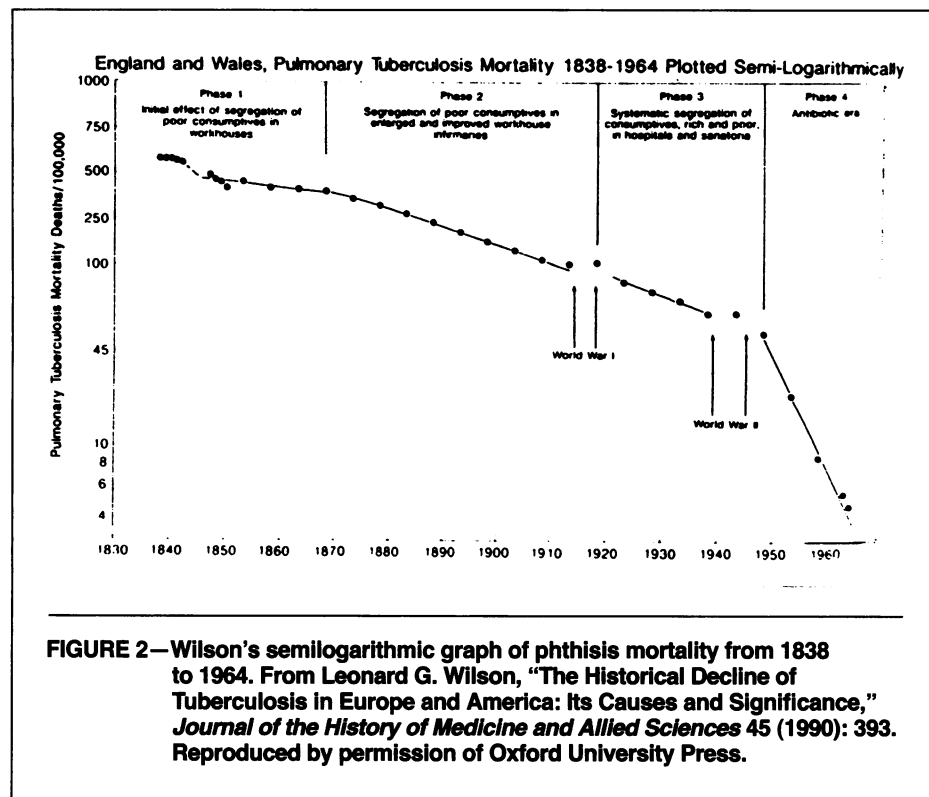
Wilson attempts to further strengthen Newsholme's thesis by analyzing secular trends in TB mortality for the period 1838 to 1961. When Wilson plotted the log of TB data for England and Wales, he observed 4 straight lines corresponding, he argues, to distinct phases in the public health program⁵⁷ (see Figure 2). As programs focused increasingly on isolation or treatment of the infected (as opposed to isolation of only the poor con-

sumptives), the rate of decline accelerated. Wilson found a similar pattern when he plotted data from New York City from 1810 to 1960.⁵⁸ He found a steeper decline in Minnesota, which by 1918 had achieved the highest rate of segregation in the nation.⁵⁹ Wilson observed that TB declined in Minnesota as a consequence of that state's efforts to isolate index cases and identify contacts, particularly family members.⁶⁰

To be sure, Wilson's argument is not unassailable. Mortality data, especially 19th-century mortality data, are incomplete.⁶¹ A decline in mortality rates, moreover, does not automatically translate into a decrease in TB incidence rates, as Wilson's analysis implies.⁶² Neither Wilson nor Newsholme had a direct measure of the rate of patient segregation. And neither controlled simultaneously for potentially confounding variables.⁶³

In addition, Wilson simplifies Newsholme's position, holding him to a mono-causal explanation for the historical fall of TB mortality rates.⁶⁴ Newsholme, however, understood that consumption was a complex disease whose decline was the consequence of multiple factors. Newsholme believed, for example, that overcrowding within dwellings, urbanization, "well-being" (determined by wages, pauperism, and total cost of living), nutrition, and education about indiscriminate spitting and coughing had an impact on the TB death toll.⁶⁵ Newsholme did not discount the importance of nutrition, wages, and housing; he simply argued that such factors lacked a predominant influence. "Segregation in general institutions is the only factor which has varied constantly with the phthisis death-rate.... It must therefore be regarded as having exerted a more powerful influence on the prevention of phthisis than any of the other factors of which none has varied constantly with the phthisis death-rate."⁶⁶

As a public health official, Newsholme was particularly concerned about practical and cost-beneficial opportunities to reduce the toll of TB. His research was meant to demonstrate the importance of pragmatic solutions to the epidemic. Poverty, overcrowding, and malnutrition, while of consequence, were not only variable, relatively weak determinants of the decline of TB but represented difficult issues for public health to confront. Institutional segregation, especially of open cases, was, to Newsholme, both efficacious and politically realistic. As he wrote in his recollections, published in 1936, "I drew satisfaction... inasmuch as the machinery to provide the segregation already existed, had been operating to a material extent for forty or fifty years, and could easily be vastly expanded at a far lower expense than could be incurred by the construction of a new machinery."⁶⁷



At the time Newsholme wrote his autobiographical recollections on public health, he was serving as advisor to a multisite health project in New York State underwritten by the Milbank Memorial Fund. One of the objectives of the project was to organize community health work to determine the cost and effectiveness of measures for the control of TB. One of the sites, Cattaraugus County, a rural county of 75 000 persons, became an epidemiologic and public health laboratory, beginning in 1923.⁶⁸ The work done in Cattaraugus County was influenced by an earlier project, the Framingham Community Health and Tuberculosis Demonstration, but generated more sophisticated statistical analyses.⁶⁹ Among the administrative procedures eventually evaluated in Cattaraugus County was the role of the sanatorium in TB control. A prospective comparative study conducted by Jean Downes found that the difference in infection rates—not mortality rates—for two groups of families was "probably associated with an improvement in the extent to which sanatoria care was obtained for the infectious cases and the speed with which they were hospitalized."⁷⁰ Significantly, the reduction in mortality was achieved during the height of the Depression.

In a later report on her research, "Salient Points of Attack against Tuberculosis," Downes offered quantitative support for using the "tuberculous family," with an infection rate 10 to 15 times that of the general community, as the locus of case finding. In the

same piece, she approvingly quoted an article by Wade Hampton Frost, the leading epidemiologist of the period, in which he outlined a program for the control of TB that emphasized "the isolation in sanatoria of all known open cases of pulmonary tuberculosis,"⁷¹ a recommendation that parallels that of Newsholme. Yet Frost, who held the first chair of epidemiology at Johns Hopkins University, observed that to effectively control TB, societies did not have to isolate all active cases.

For the eventual eradication of tuberculosis it is not necessary that transmission be immediately and completely prevented. It is necessary only that the rate of transmission be held permanently below the level at which a given number of infection spreading (i.e. open) cases succeed in establishing an equivalent number to carry on the succession. If, in successive periods of time, the number of infectious hosts is continuously reduced, the end-result of this diminishing ratio, if continued long enough, must be the extermination of the tubercle bacillus.⁷²

Downes' findings and Frost's strictures provide support, based on contemporary data, for Newsholme's retrospective analysis. Isolation of active cases of TB reduces the incidence of secondary attacks on those living in close proximity to the index patients; over time, such isolation reduces the prevalence of the tubercle bacillus and, subsequently, TB incidence and mortality rates in the population as a whole. In brief, although contemporary historians have often denied the value of segregation, both original data and the quan-

titative analyses of earlier historians strongly suggest that patient isolation did make a difference.

Bovine Tuberculosis

While the sanatorium has figured significantly in numerous histories of tuberculosis, other bacteriologically based methods for its control have received short shrift in the historical literature. Among these are pasteurization, tuberculin testing, and eradication efforts among cattle. The silence of historians, however, should not be interpreted as a commentary on the effectiveness of these measures. Such measures constituted significant, successful public health initiatives, interventions that produced scientific, economic, and political controversies that have since been forgotten.⁷³

The association between a slow, wasting disease in cattle and phthisis in humans was suspected for centuries before Koch's discovery of the human tubercle bacillus in 1882.⁷⁴ Sixteen years later, Theobald Smith isolated a bovine mycobacterium, implicating it as the major determinant of TB in cattle. Based on differences between the 2 microbes and his inability to transmit human TB bacilli to cattle, Koch concluded that bovine TB posed little threat to humans.⁷⁵ His position generated considerable dissension from veterinarians and led to the appointment of government-sponsored commissions in Germany, Britain, and New York City. In reports issued between 1904 and 1911, all concluded that *Mycobacterium bovis* was a source of human TB.⁷⁶

Epidemiologic studies representing varying places and times indicate that bovine-induced TB occurs in humans, although at a rate far lower than TB due to *M tuberculosis*, and is associated primarily with nonpulmonary infections. For the period from 1911 to 1927, for example, studies found that the overall percentages of TB cases due to bovine strains in the areas around Cambridge, England, were approximately 23% for all forms of TB and approximately 1% for pulmonary TB (Bryder estimates 30% for nonpulmonary TB and 2% for pulmonary TB in England during the 1930s⁷⁷). However, among children 5 years of age and younger, 68% of cases studied were attributable to bovine TB. Researchers in New York City reported a similar rate in children (66%) in 1910.⁷⁸ The Cambridge regional studies showed that the percentage of cases due to bovine TB fell with increasing age: 37% among children 5 to 15 years old and 6% in those 15 years of age and older.⁷⁹ In general, the prevalence of TB was higher in rural areas of England and Scotland, with overall rates of bovine (and human) TB 300% greater than

those in the Cambridge area.⁸⁰

In the countries of North America and western Europe, the growing suspicion of an association between TB in cattle and humans precipitated social policy responses. In the United States, the passage of the Meat Inspection Act of 1906 made inspection of carcasses for TB mandatory across the nation. Yet meat inspection neither eliminated the source of TB nor aided cattlemen. The increasing rate of TB in cattle threatened the nation's livestock industry. One expert estimated that the number of cattle condemned as unfit for human consumption in 1917 could fill a train 15 miles long. In Chicago in 1916, the packing houses, stockyards, railroads, and stock exchange organized a committee and provided funding to eliminate TB in cattle and hogs. The following year, the Tuberculosis Eradication Division of the US Department of Agriculture's Bureau of Animal Industry spearheaded a long-term TB eradication program that combined tuberculin testing of cattle, eradication of all reacting animals with compensation to farmers for their losses, certification of "Tuberculosis Free Accredited Herds," and establishment of "Modified Accredited Tuberculosis Free Areas."⁸¹

Although the division's program did not begin until 1917, the results were dramatic. Figure 3 compares the rate of tuberculin reactors found and removed from cattle populations with TB mortality in the general population and among several subgroups. Without exception, the initiation of the federal TB cattle eradication program (1917) and the peak in the detection and removal of infectious cattle (1918) coincide with sharp declines in pulmonary and extrapulmonary TB mortality. It is unlikely that the cattle eradication program was solely responsible for the decline in TB mortality, especially given that its successes did not precede the decline in human mortality.⁸² The period from 1915 to 1921 was one of rapid spread of pasteurization in American cities.⁸³ Together, pasteurization and testing of cattle probably played a major part in the accelerated decline in extrapulmonary and perhaps even pulmonary TB, beginning in 1917.

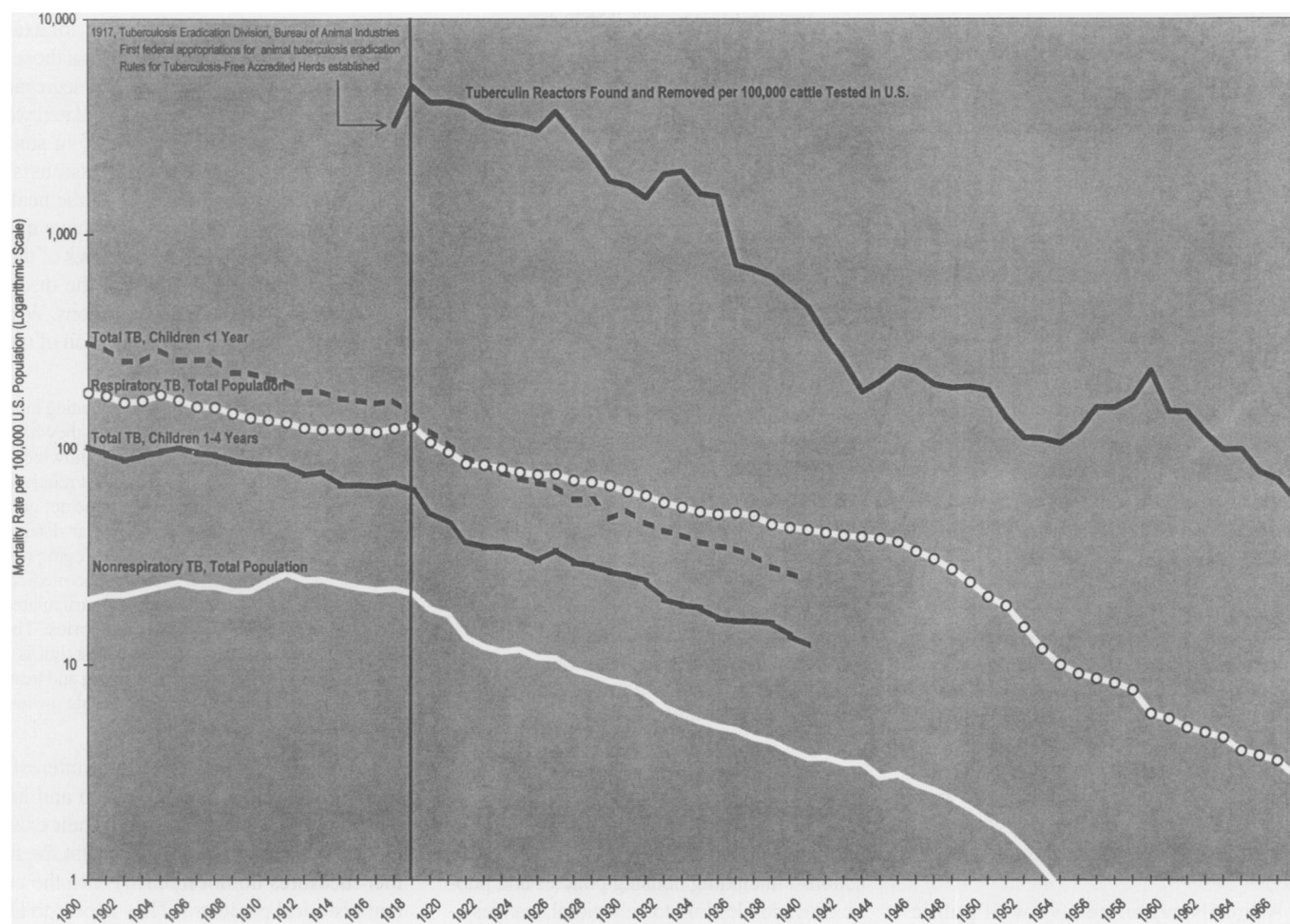
Significantly, in the period after 1940, the resurgence of TB among cattle seems to have precipitated an increase in extrapulmonary TB in the general population. By 1940, every US county had been officially designated a "Modified Accredited Tuberculosis Free Area," meaning that the number of tuberculin reactors among cattle did not exceed 0.5% and that the county was in compliance with federal testing and disposal rules.⁸⁴ Many cattle owners and legislators regarded this veterinary milestone as the final goal, and tuberculin testing among cattle consequently decreased. The rate of tuberculin reactors

found among cattle continued to decline, reaching an all-time low in 1952. In 1955, however, the incidence of TB among livestock began to rise. By 1959, it had increased to 23 per 10 000 animals tested. As Myers and Steele, who documented the history of the control of bovine TB, concluded, "Resumption of the long time standard program of extensive periodic tuberculin testing again proved its efficacy and the incidence of infection decreased from 23 per 10,000 in 1959 to 6 per 10,000 in 1969."⁸⁵ Contemporary epidemiologists recognize the need for continued close surveillance of animal and human reservoirs for *M bovis* TB.

In the early years of the 20th century, localities in the United States also instituted pasteurization of milk, with the aim of eliminating milk as a vehicle for scarlet fever, diphtheria, infant diarrhea, and TB.⁸⁶ In 1909, the US Public Health Service reported 500 epidemics of milkborne disease in the period 1880 to 1907.⁸⁷ That year, Chicago's health commissioner, unable to regulate the 12 000 dairies that supplied the city's residents with milk, promulgated a compulsory pasteurization ordinance, the first in the United States.⁸⁸ The following year, New York City required that all milk for drinking purposes be pasteurized.⁸⁹ By 1922, 90% or more of milk sold in most large US cities was pasteurized.⁹⁰

Where pasteurization was introduced, tuberculosis declined, particularly among children.⁹¹ In Toronto, which prohibited the sale of nonpasteurized milk beginning in 1918, bovine TB disappeared as an admitting diagnosis in the local children's hospital for any child born or raised in the city.⁹² The introduction of pasteurized milk in the Netherlands in 1940 led to a marked decline in TB.⁹³ With the elimination of milk as the principal vector of transmission of bovine TB, the anatomical distribution of TB lesions also changed dramatically. Meningeal, bone, joint, and skin TB, previously very common, became rare.⁹⁴

Western nations that resisted the elimination of tuberculous cattle or the adoption of pasteurization saw TB rates decline more slowly, particularly in children. In Britain, as both Bryder and Smith explain, farmers, consumers, and legislators repeatedly resisted attempts to make tuberculin testing of cattle and pasteurization of milk compulsory. The financial burden of tuberculin testing, the subsequent loss of infected cattle, and concerns regarding the safety, taste, and price of pasteurized milk combined with the scientific controversies to effectively block enforceable national safety standards. Not until the 1950s was meat inspection uniform, and not until after 1948 was pasteurization compulsory.⁹⁵ Most of the decline in nonpulmonary TB mortality in Britain occurred in the decade following the outbreak of World War II, once



Source. J. Arthur Meyers and James H. Steele, *Bovine Tuberculosis Control in Man and Animals* (St. Louis, Mo.: Warren H. Green, 1969); Vital Statistics of the United States; World Health Organization, Annual Epidemiological and Vital Statistics and World Health Statistics Report.

FIGURE 3—Bovine TB eradication and human TB mortality rates, United States, 1900 to 1967.

pasteurization spread quickly across the country.⁹⁶ Nonetheless, in 1951, the rate of tuberculosis attributable to *M bovis* was higher in Britain than in any other industrial nation.⁹⁷ The British conclusion, then, is that an opportunity to prevent disease was practically wasted: "Pasteurisation saved lives and could have saved them earlier."⁹⁸

Conclusion: History as Politics

What caused the decline in TB mortality? Those who have attempted to answer that question often have a set of political values that informs their answers. McKeown, whose work continues to frame so much of the debate over TB mortality, had lost faith in clinical interventions early in his career. His own training in and experience of medicine left him convinced that its usefulness was greatly exaggerated.⁹⁹ As a professor of social medicine

at Birmingham University during the post-World War II years, when Britain was establishing its National Health Service, he fought against those who would have had the Service overcommit to traditional secondary prevention.¹⁰⁰ He argued that the Service must promote major efforts to modify environmental factors and personal behaviors, "which are the predominant determinants of health."¹⁰¹ For McKeown, history was an appropriate tool for attacking medicine; the social history of medicine, he noted, "is essentially an operational approach which takes its terms of reference from difficulties confronting medicine in the present day."¹⁰²

At the same time that he deliberately attacked medicine, he shared with other academic members of his generation a certain disdain for public health. According to Szreter, "Social medicine provided an altogether grander vision of positive health enhancement

for the populace through deployment of the resources and organization of the state."¹⁰³ Indeed, social medicine not only represented a new view of health and medicine but amounted to a "rejection of the narrow focus and complacency of public health."¹⁰⁴ For McKeown, public health was a limited, obsolete method of affecting health, using local administrative apparatus dating from the Victorian era to put into place various technologies that reduced environmental pathogens.¹⁰⁵ A certain air of whimsical condescension is evident in his autobiographical account describing his ascendance to the chair of social medicine:

I eventually took possession of a room in the Medical School, shared previously by the part-time teacher of public health (Dr. G. A. Auden, father of the poet) and the lecturer in forensic medicine, and filled with the drains, waterpipes, contraceptives, and numerous other



HOME "FINISHERS": A CONSUMPTIVE MOTHER AND HER TWO CHILDREN

Both of the children work and sleep with the mother.

This image of a tubercular woman and her children at work in a tenement underscores the potential role both of fundamental sanitary and housing reform and of isolation of active cases in staying the spread of tuberculosis. From Sprague, *The Bitter Cry of Children* (New York: Macmillan, 1906).

objects of forensic and nineteenth century public health interest.¹⁰⁶

Arthur Newsholme's view of public health, on the other hand, was that of a progressive Victorian medical officer of health deeply influenced by Koch's discovery of the tubercle bacillus and the doctrine of TB as a contagious disease.¹⁰⁷ Like other medical officers of health, such as Arthur Ransome and James Niven, Newsholme supported and proposed measures to reduce the transmission of consumption. He made Brighton, where he served as medical officer of health, the first town that officially established voluntary reporting of consumption, in 1899.¹⁰⁸ In this endeavor, he faced formidable political and social obstacles in a society in which TB was a stigmatizing disease and the ethical and practical effects of isolating active cases were seriously debated.¹⁰⁹ Nonetheless, as medical officer of the Local Government Board, Newsholme successfully introduced compulsory notification of TB as a national policy, gradually implementing it beginning in 1908.¹¹⁰

Newsholme's campaign to isolate active TB cases was of a piece with his belief in compulsory notification; his epidemiological analyses provided, he believed, scientific support for his position that segregation was an effective, affordable measure against consumption. For Newsholme and other medical

officers of health, targeted public health actions—including housing policies and public education leading to behavioral changes—could effectively contribute toward the decline in TB incidence and mortality; such limited interventions were, moreover, politically feasible, unlike broader social reform affecting nutrition and poverty.¹¹¹

Like McKeown, many historians of medicine, especially those affected by the politics of the 1960s and 1970s, have been skeptical of the efficacy claims made by the medical profession and—by extension—the "new" public health, which produced a new cadre of public health professionals,¹¹² justified a new set of interventions, and, increasingly, overlapped with clinical medicine.¹¹³ They have often reacted strongly against the beliefs of previous generations of historians, who tended to celebrate the successes of physicians, to support the rational authority of biomedicine, and to hold to a faith in the historical progress of medical science.¹¹⁴ Instead of the efficacy of biomedicine, they have tended to concentrate on "the social causation of health and disease and the way in which science is embedded in a society's social relations."¹¹⁵

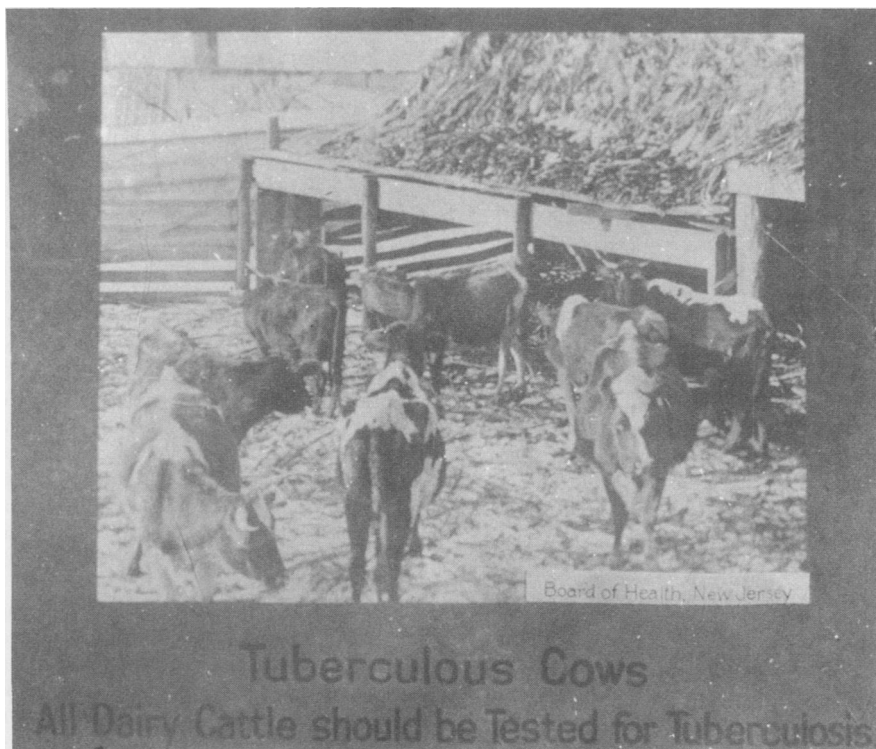
Influenced by the civil rights, anti-Vietnam War, women's liberation, and gay liberation movements, these medical historians have often focused their attention on women, minorities, and the poor within the health care

system. Recognizing that medicine and medical institutions could be a means of social control, they have been suspicious, for example, of public health actions such as those of Newsholme and his generation that segregated the sick, particularly when those isolated were poor or otherwise dispossessed.¹¹⁶ In studying the history of medical professions and institutions, including those of public health, contemporary medical historians have questioned the social and political motives of those involved and the objectivity of the disease entities that often justified their actions. Writes Charles Rosenberg, a leading historian of medicine:

Critics have turned the delegitimizing tools of cultural relativism on medicine as they have on so many other areas in which knowledge and power are closely linked.... This relativist point of view has sought to undermine not only the apparent objectivity of particular disease entities but also, by implication, the legitimacy of the social authority wielded by the medical profession, which has traditionally articulated and administered diagnostic categories. The physician is not above social interest, but is a social actor whose mission of defining and treating disease can express and legitimate professional, class, or gender interests.¹¹⁷

The reason for the dearth of interest of social historians in pasteurization and herd eradication, in sharp contrast to their examination of sanatoriums, may be that the former measures do not fit easily into the current historical paradigm. They appear to lack the political, social, and cultural dimensions that make interventions such as segregation of the infected provocative. Issues of the relative roles of clinical medicine and public health, class dissension, social control of knowledge, or construction of disease seem irrelevant here. Measures such as pasteurization and tuberculin testing seem minor, rather technical themes relevant to histories of veterinary science, the spread of technology, or the milk industry, none of which are of much interest to contemporary social historians of medicine. The story of pasteurization and herd eradication, at least in its current telling, is closer to the older history of technical progress and success than to the new social history.

In the essay already cited, Charles Rosenberg argues that the HIV/AIDS epidemic has served notice to society that some diseases—including cholera and TB—are distinctly biological as well as social phenomena.¹¹⁸ Sophisticated immunological and virological techniques were required to define HIV/AIDS as a clinical entity and to develop chemotherapies to treat it. Although AIDS and the response to it must be placed within a complex cultural context, the biomedical dimensions of that disease must be recognized as having



Lantern slide warning of tubercular cows, New Jersey. Courtesy of the State Historical Society of Wisconsin, Visual Materials Archive.

profound scientific, clinical, and policy consequences.¹¹⁹

AIDS policy successes provide a strong reason to revisit the role of public health during past disease episodes. Faced with a new disease for which cure was unknown and treatment limited or unavailable, public health professionals cobbled together policies that, without eliminating AIDS, probably reduced the risk of HIV infection and its sequelae. These targeted public health interventions include heat treatment of blood products, blood donor screening, and the institution of needle exchange programs. Such policies, each of which generated opposition, make us more sensitive to the efficacy of small victories, as well as the complexities of an incurable infectious disease. Consequently, they affirm, in an analogous fashion, that targeted interventions incrementally decreased the incidence of TB. In brief, current experiences during the HIV/AIDS epidemic may have served to produce, if not a revolution, then a shift in the current medical history paradigm.

This review of historical literature on TB, we argue, strongly suggests the need for a careful assessment of the role of public health interventions in the decline of TB morbidity and mortality. It supports the hypothesis that public health interventions had an impact on the falling rates of mortality from infectious

diseases in general and TB in particular, beginning in the late 19th century. Certainly, public health alone could not have effected this change. Changes in sources of immigration, for example, may have decreased the incidence of TB in Minnesota in the late 19th and early 20th centuries.¹²⁰ Improvements in nutrition and a rise in the general population's social conditions may also have contributed to the decline of TB mortality. In the 20th century, clinical interventions such as chemotherapy played a substantial role in the drop in TB's toll.

As Nancy Tomes observes, McKeown's work continues to reinforce scholarly tendencies to dismiss all but the broadest social forces.¹²¹ Within the literature we have reviewed here, the reverse is also true: many attacking McKeown, bent on toppling an icon, tend to focus on only the narrowest public health interventions.¹²² Moreover, figures such as Newsholme and public health organizations such as the Milbank Memorial Fund were not simply neutral commentators but, rather, agents of public health who favored relatively easily engineered targeted interventions—"drawing a bow at a venture and aiming straight at the mark"¹²³—over politically and technically complex social interventions that would affect a population much wider than infected individuals and their

immediate contacts.

The findings of Bayer et al., presented in this issue of the Journal, underscore the way in which a relatively easily engineered targeted intervention can produce impressive results.¹²⁴ Directly observed therapy played a crucial role in improving the effectiveness of TB drug treatment in cities that had fared miserably before 1993, prior to an influx in federal funding for such therapy. Yet success sparks the risk that advocacy for a targeted intervention can take on the qualities of an orthodoxy resistant to a more complex perspective. Bayer et al. also found that locales that had achieved high treatment completion rates without universal directly observed therapy improved those rates only marginally, and at very high costs. However, those who had advocated for directly observed therapy as a solution were loath to acknowledge that anything less than universal directly observed therapy could achieve the desired public health results. Accordingly, the work by Bayer et al. developed a complex history of its own as it traveled from one major medical journal to the next, receiving starkly mixed reviews that foreshadowed a storm of controversy. Understanding that institutional commitments often drive methodological critiques that undercut heterodox findings, one anonymous reviewer equated DOT (directly observed therapy) with "Dogma on Tuberculosis."

On the one hand, then, we urge that a nuanced historical and epidemiological approach to the question of TB be restored. If the relative contribution of different interventions and factors is to be sorted out, pursuit of monocausal explanations for the retreat of TB, like monotypic intervention, is insufficient. On the other hand, while we might call for more complexity in both debate and scholarship, our review suggests that this will be problematic. In addition to the institutional, ideological, and political commitments that individuals bring to their analyses and arguments, we are left with the harsh reality that TB was and is a disease inequitably distributed in society; immigrants, the poor, the homeless, and individuals infected with HIV are its primary carriers.¹²⁵

Targeted interventions are Janus-faced: by definition, they visit upon the sick the burdens of intervention; at the same time, those very burdens can effectively reduce the toll of disease. And herein lies the fundamental dilemma: in bringing together history, policy, and advocacy in the case of TB, we have created a false choice between broad social change and one or another targeted public health intervention, between nihilism and pragmatism. It is not only the case, then, that we must bring a diversity of methods

and evidence that embrace the specificity of time, place, and person to bear on the problem of TB. We must also acknowledge the fundamentally political nature of the problem and of the solutions we offer as historians and policy advocates. □

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- Ronald Bayer and David Wilkinson, "Directly Observed Therapy for Tuberculosis: History of an Idea," *Lancet* 345 (June 17, 1995): 1545.
- Money available to the Centers for Disease Control and Prevention for TB control rose from \$25 million in 1991 to \$104 million in 1993. Bayer and Wilkinson, "Directly Observed Therapy," 1547.
- Ibid.*
- Ibid.*, 1545–1548; C. Patrick Chaulk et al., "Eleven Years of Community-Based Directly Observed Therapy for Tuberculosis," *Journal of the American Medical Association* 274 (1995): 945–951; S. E. Weis et al., "The Effect of Directly Observed Therapy on the Rates of Drug Resistance and Relapse in Tuberculosis," *New England Journal of Medicine* 330 (1994): 1179–1184; R. L. Hotchkiss, "Directly Observed Treatment of Tuberculosis," *New England Journal of Medicine* 329 (1993): 135.
- Interestingly, when discussed in isolation, directly observed therapy was not disavowed in favor of broader social interventions involving housing and institutions. Rather, it was initially opposed as too costly and labor-intensive. Among policymakers striving to devise immediate, pragmatic policy solutions, directly observed therapy was challenged as an intrusion on autonomy, as a violation of the constitutional requirement of due process and the requirement that the least restrictive alternative be used, and as contrary to legal requirements that restricting the behavior of individuals with disabilities be based on individualized assessments. Bayer and Dupuis, "Tuberculosis, Public Health, and Civil Liberties," 318–319; "Appendix A: Dissents to Recommendations," in *The Tuberculosis Revival: Individual Rights and Societal Obligations in a Time of AIDS* (New York: United Hospital Fund, 1992) 30–32; George J. Annas, "Control of Tuberculosis—The Law and the Public's Health," *New England Journal of Medicine* 328 (1993): 587–588.
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- See McKeown, *The Role of Medicine*, 33; McKeown calculated that 40% of the reduction in mortality between 1848 and 1971 was due to a decline in airborne diseases; 21%, to water- and foodborne diseases; 13%, to other conditions; and 26%, to a decrease in conditions not associated with microorganisms.
- This critique of the McKeown thesis is from Simon Szreter, "The Importance of Social Intervention in Britain's Mortality Decline c. 1850–1914: A Re-interpretation of the Role of Public Health," *The Society for the Social History of Medicine* 1 (1988): 7–10.
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- Ibid.*, 59–65.
- Szreter, "Social Intervention," 13, 16–17. Using McKeown's own data, Szreter argues that rising bronchitis rates in the second half of the 19th century weaken McKeown's assertion that the fall of respiratory rates in general or TB rates in particular was critical to the decline in that cen-

ture's mortality rates. Szreter also suggests that a sustained decline in TB rates did not occur until after 1867. He then argues:

Improvement in respiratory TB would, then, no longer appear to have been either the chronologically prior or the quantitatively predominant feature of the nineteenth century mortality decline in England and Wales. According to the logic of McKeown's own arguments, the foregoing would indicate a primary role for sanitary reform and public health measures, rather than rising nutritional levels or living standards. The changing incidence of mortality from respiratory TB in Victorian Britain, rather than being cast in the role of a leading and determining influence, can be seen as a dependent function of the general intensity and frequency of other debilitating diseases. (16–17)

For further discussion of the relationship between intercurrent infections and TB, see Alex Mercer, *Disease, Mortality and Population in Transition, Epidemiological-Demographic Change in England since the Eighteenth Century as Part of a Global Phenomenon* (Leicester: Leicester University Press, 1990), 97–123.

24. Szreter, "Social Intervention," 13.
25. Ibid. For other studies arguing that the public health movement in Great Britain was a significant cause of the general mortality decline, see Anthony S. Wohl, *Endangered Lives: Public Health in Victorian Britain* (Cambridge, Mass.: Harvard University Press, 1983); George Rosen, *A History of Public Health* (New York: MD Publications, 1958); G. T. Griffith, *Population Problems in the Age of Malthus* (New York: Cambridge University Press, 1926); M. C. Buer, *Health, Wealth and Population in the Early Days of the Industrial Revolution* (London: Routledge, 1926); P. E. Razzell, "An Interpretation of the Modern Rise of Population in Europe: A Critique," *Population Studies* 28 (1974): 5–17; and J. C. Riley, *The Eighteenth-Century Campaign to Avoid Disease* (London: Macmillan, 1987).
26. Local studies include Anne Hardy, *The Epidemic Streets: Infectious Disease and the Rise of Preventive Medicine, 1856–1900* (Oxford, England: Clarendon Press, 1993); Neil McFarlane, "Hospitals, Housing, and Tuberculosis in Glasgow," *Social History of Medicine* 2 (1989): 59–85; and Gillian Cronjé, "Tuberculosis and Mortality Decline in England and Wales, 1851–1910," in *Urban Disease and Mortality in Nineteenth Century England*, ed. Robert Woods and John Woodward (London and New York: Batsford Academic and Educational and St. Martin's Press, 1984).
27. Hardy, *Epidemic Streets*, 249–250, 265.
28. McFarlane, "Hospitals, Housing, and Tuberculosis," 59.
29. Curtin finds that the greatest mortality gains between 1839 and 1870 were overseas, where targeted efforts to improve sanitary conditions were implemented. Over the next 20 years, as sanitary engineering became more common in Europe, the greatest gains in mortality took place at home. Philip Curtin, *Death by Migration: Europe's Encounter with the Tropical World in the Nineteenth Century* (Cambridge, England: Cambridge University Press, 1989): 80, 159.
30. Between 1837 and 1846, TB caused 50% of all deaths among the troops in Britain. During the 1860s, it caused 38% of the deaths of British troops and 22% of the deaths of French troops posted in their respective countries. However, it contributed to only 7% of all deaths among troops in Madras, Algeria, and the Windward and Leeward islands during the 1840s. Curtin, *Death by Migration*, 76. In the 1820s and 1830s, less than 10% of deaths among British troops abroad were attributable to TB, as compared with 55% of deaths among British troops in England. In the period from the 1840s to the 1860s, although TB mortality dropped 75% among troops in Britain, the mortality rate from TB continued to be higher in Britain (63.38 deaths per 1000 troops in Britain vs 7.53 deaths per 1000 in the Windward and Leeward Command and 6.99 per 1000 in Madras for the period 1837 to 1847; 3.16 deaths per 1000 troops in Britain vs 1.6 per 1000 in the Windward and Leeward Command and 2.86 per 1000 in Madras for the period 1859 to 1867). Curtin, *Death by Migration*, 30, 35.
31. Curtin, *Death by Migration*, 42, 76. See also S. Guha, "Nutrition, Sanitation, Hygiene and the Likelihood of Death: The British Army in India," *Population Studies* 47 (1993): 385–401.
32. Mitchell, "Inexact Science," 387.
33. Nancy Tomes, "The White Plague Revisited," *Bulletin of the History of Medicine* 63 (1989): 467, 468.
34. Linda Bryder, *Below the Magic Mountain: A Social History of Tuberculosis in Twentieth-Century Britain* (Oxford, England: Clarendon Press, 1988), 30; Barbara Bates, *Bargaining for Life: A Social History of Tuberculosis, 1876–1938* (Philadelphia: University of Pennsylvania Press, 1992).
35. See also Lerner, "New York City's Tuberculosis Control Efforts," 758–766.
36. The beginning of the decline in TB mortality in England is highly contested. McKeown argues that the decline began in the 1830s, if not earlier, whereas Szreter counters that the decline did not begin before 1867, if not later. Anne Hardy supports Szreter's figures and writes that "the beginning of the decline may be pushed even further forward, towards the 1880s." Hardy, *Epidemic Streets*, 214. Scholars consistently maintain that the decline in the United States began in the 1870s, although there are no data comparable to the Registrar General's mortality statistics for England and Wales supporting this estimate.
37. The overall annual mortality rate from TB in 1900 was approximately 200 per 100 000 population in the United States. Among Blacks, the rate was 400 per 100 000. Barbara Gutmann Rosenkrantz, "Introductory Essay: Dubos and Tuberculosis, Master Teachers," in Rene Dubos and Jean Dubos, *The White Plague: Tuberculosis, Man, and Society* (New Brunswick, N.J.: Rutgers University Press, 1952, 1987), xiv–xv, note 1. In urban areas, the mortality rate was higher. In New York City, for example, the mortality rates were 428 per 100 000 in 1870 and 256 per 100 000 in 1890. The New York City mortality rate varied from 49 per 100 000 on the upper West Side to 776 per 100 000 in the tenement district of lower Manhattan. Sheila M. Rothman, *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in American History* (New York: Basic Books, 1994), 184. In Philadelphia, the mortality rate in 1870 was 350 per 100 000; this rate had dropped to less than 200 per 100 000 by 1910. By 1925, the mortality rate was less than 100 per 100 000. Bates, *Bargaining for Life*, 315.
- In Pennsylvania, the TB mortality rate first dropped to below 10 deaths per 100 000 in 1956. In England and Wales, for all forms of TB, the mortality rate was close to 400 per 100 000 in 1865; it declined to less than 200 per 100 000 in the decade before 1900 and to 100 per 100 000 in 1930. Bryder, *Below the Magic Mountain*, 7. The vast majority of cases (98% of all pulmonary cases and 70% of nonpulmonary cases) were due to infection with the human TB bacillus. The remaining 2% of pulmonary cases and 30% of nonpulmonary cases were attributable to bovine infection caused by infected meats and, more often, infected milk. Bryder, *Below the Magic Mountain*, 3.
38. In Great Britain, there were approximately 17 hospitals for the treatment of TB (representing about 1100 beds) by 1893. By 1907, there were 96 institutions for the "open-air treatment" of TB (4081 beds). Not counting Poor Law beds, there were, in 1911, 5500 beds in 259 local and voluntary sanatoriums and isolation hospitals (97 local institutions, 79 voluntary institutions, and 83 isolation hospitals; 1300 beds were provided by local authorities and 4200 by voluntary institutions). By 1916, the number of beds had increased to nearly 12 000. Of the 11 893 beds, 6072 were provided by local authorities and 5821 by voluntary institutions. In 1920, there were 145 781 beds, 8845 provided by local authorities and 6936 by voluntary institutions. Bryder, *Below the Magic Mountain*, 23, 32, 44. In the United States, in comparison, there were 34 sanatoriums (representing 4485 beds) in 1900; by 1925, there were 536 sanatoriums with more than 700 000 beds. Rothman, *Shadow of Death*, 198. In Pennsylvania—the state with the most extensive system of TB institutions—there were approximately 2000 beds by 1910 and slightly less than 4000 by 1920. Bates, *Bargaining for Life*, 281.
39. Bryder, *Below the Magic Mountain*, 24.
40. Ibid., 36–41.
41. Ibid., 68–69.
42. F. B. Smith, *The Retreat of Tuberculosis, 1850–1950* (London: Croom Helm, 1988), 244–245.
43. Sheila M. Rothman, "The Sanatorium Experience: Myths and Realities," in *The Tuberculosis Revival: Individual Rights and Societal Obligations in a Time of AIDS* (New York: United Hospital Fund, 1992); 72.
44. Rothman, *Shadow of Death*, 209.
45. Rothman, "Sanatorium Experience," 73.
46. Regarding the TB control movement as a whole, Bates similarly asserts that "[n]o effects of the tuberculosis movement can be seen in the falling mortality rates at least until the 1920s and, even then, they are not clear." Bates, *Bargaining for Life*, 331.
47. Ibid., chaps. 2, 5, and 17.
48. Bates observes that the proportion of patients dying in sanatoriums increased from 2% to more than 18% from 1909 to 1916. Bates, *Bargaining for Life*, 165–166.
49. Ibid., 157–158.
50. Ibid., 318.

51. Ibid., graph 17-2, 319.
52. Ibid., 318, 321-325. Among the other causes, Bates identifies an increase in per capita income, improvements in ventilation in a wide range of institutions, improvements in the conditions and speed of immigrant transatlantic travel, and nutritional advances that would have come hand in hand with innovations in preservation and transportation.
53. Tomes points out that, although their publications conveniently dovetailed with the resurgence of TB, scholars turned their attention to the social history of TB well before the resurgence gained the attention of public health officials and policymakers. Tomes, "White Plague Revisited," 467. Although these works, which were published after policy debate over how to control the resurgence of TB began, resonate with contemporary and historical debate over the retreat of the disease, they were neither inspired nor driven by such debate.
54. Leonard G. Wilson, "The Historical Decline of Tuberculosis in America: Its Causes and Significance," *Journal of the History of Medicine and Allied Sciences* 45 (1990): 366-396; Samuel H. Preston, *Mortality Patterns in National Populations* (New York: Academic Press, 1976).
55. In 1911, more than 60% of TB beds in Britain were located in Poor Law infirmaries.
56. Specifically, the Irish Poor Law of 1847, passed in the wake of the potato famine, prohibited wandering abroad, moving from one locale to another, or begging in public. Newsholme, "Poverty and Disease, as Illustrated by the Course of Typhus Fever and Phthisis in Ireland," *Epidemiological Section, Proceedings of the Royal Society of Medicine* 1 (1907-1908): 14.
57. An exponential phenomenon plotted logarithmically will produce a straight line, the slope of which corresponds to the rate of change.
58. Wilson, "Decline of Tuberculosis," 389-395.
59. Leonard G. Wilson, "The Rise and Fall of Tuberculosis in Minnesota: The Role of Infection," *Bulletin of the History of Medicine* 66 (1992): 16-52.
60. Ibid., 50.
61. Linda Bryder, "'Not Always One and the Same Thing': The Registration of Tuberculosis Deaths in Britain, 1900-1950," *Social History of Medicine* 9 (August 1996): 253-265.
62. Neither Newsholme nor Wilson took into account the lag time between an intervention (segregation) and the decline in TB-specific mortality rates as a consequence of reduced infection incidence rates. In addition, mortality rates are a function of incidence and case fatality rates during the periods in question; better care might conceivably have reduced the case fatality rate.
63. For further criticism of Newsholme's studies, see John M. Eyler, *Sir Arthur Newsholme and State Medicine, 1885-1935* (Cambridge, England: Cambridge University Press, 1997): 182-186.
64. Wilson, "Decline of Tuberculosis," 367.
65. Newsholme, "The Notification of Phthisis Pulmonalis," *The Practitioner* 67 (1901): 27; Newsholme, "An Inquiry into the Principal Causes of the Reduction in the Death-Rate from Phthisis during the Last Forty Years, with Special Reference to the Segregation of Phthisical Patients in General Institutions," *Journal of Hygiene* 6 (July 1906): 320; Newsholme, *The Last Thirty Years in Public Health: Recollections and Reflections on My Official and Post-Official Life* (London: George Allen & Unwin, 1936): 130-131. Newsholme also held that improved sanitation (including drainage and improved ventilation and lighting) and improvement in the milk supply interdicted the transmission of the tubercle bacillus. Newsholme, "An Inquiry into the Principal Causes," 350-353; Newsholme, "The Relative Importance of the Constituent Factors Involved in the Control of Pulmonary Tuberculosis," *Transactions of the Epidemiological Society of London* 25 (1905-1906): 39.
66. Newsholme, "An Inquiry into the Principal Causes," 374, 375. Significantly, while Newsholme touted the benefits of segregation, he did not credit sanatoriums themselves or knowledge of the infectiousness of TB with the retreat of the disease: "The decrease of phthisis where it has occurred cannot have been due to improved education as to the infectivity of the disease or to the introduction of Sanatoria, both of these having occurred after well-marked decrease had set in, and the Sanatoria up to the present time having been insignificant in number relative to the amount of the disease."
67. Newsholme, *The Last Thirty Years in Public Health*, 127.
68. For example, see Edgar Sydenstricker, "The Decline in the Tuberculosis Death Rate in Cattaraugus County," *Milbank Memorial Fund Quarterly* 61 (1928): 41-50; Jean Downes, "A Study of the Effectiveness of Certain Administrative Procedures in Tuberculosis Control," *Milbank Memorial Fund Quarterly* 14 (1936): 317-327; Downes, "The Risk of Mortality among Offspring of Tuberculosis Patients in a Rural Area in the Nineteenth Century," *American Journal of Hygiene* 26 (1937): 557-569; Downes, "The Effect of Tuberculosis on the Size of Family," *Milbank Memorial Fund Quarterly* 17 (July 1939): 274-287; Downes, "Salient Points of Attack against Tuberculosis," *Milbank Memorial Fund Quarterly* 18 (1940): 44-60.
- Cattaraugus County was of particular interest to investigators. Despite steady declines in the TB mortality rate in New York State as a whole and in comparable rural counties amounting to a 32% decrease between 1913 and 1923, the mortality rate of Cattaraugus County had remained constant since about 1913. *Milbank Memorial Fund Quarterly* 2 (April 1924): 1. The Milbank Memorial Fund selected "typical" American communities "to demonstrate... whether by intensive application of known health measures the extent of sickness in the United States can be further and materially diminished and mortality rates further and substantially reduced, and whether or not such practical results can be achieved in a relatively short period of time and at a per capita cost which communities will willingly bear." Quoted in C. E. A. Winslow, *Health on the Farm and in the Village: A Review and Evaluation of the Cattaraugus County Health Demonstration with Special Reference to Its Lessons for Other Rural Areas* (New York: Macmillan, 1931). See also "Controlling Tuberculosis in Cattaraugus County," *Milbank Memorial Fund Quarterly* 2 (January 1925): 5.
69. The Framingham study sought to demonstrate that public health interventions such as case finding, clinical treatment, public health nursing, and health education reduced TB-specific mortality over time. *Framingham Monograph No. 10. General Series IV. Final Summary Report, 1917-1923 Inclusive* (Framingham, Mass.: National Tuberculosis Association, 1924).
70. Downes, "Effectiveness of Certain Administrative Procedures," 323. Specifically, Downes found a 15% to 25% reduction in infection rates among families of individuals with TB who received sanatorium care from 1931 to 1935 relative to a group of families followed from 1923 to 1930, before implementation of the sanatorium program. Downes concluded: "The difference in the infection rate for the two groups of families (20.0 ± 4.56) is more than four times its probable error and may be considered as statistically significant since such a difference will arise by chance less often than once in a hundred times." Downes, "Effectiveness of Certain Administrative Procedures," 322-323.
71. Downes, "Salient Points of Attack," 59.
72. Wade Hampton Frost, "How Much Control of Tuberculosis?" *American Journal of Public Health and The Nation's Health* 27 (1937): 763.
73. The turn-of-the-century American bacteriologist who confirmed German findings that tuberculin could be used to detect TB in cattle saw little scientific consensus when it came to bovine TB: "There is scarcely a subject related to agriculture or public health that has occasioned as much or as bitter discussion, or has led to the expression of so many divergent views as this one of tuberculosis in cattle." Quoted in J. Arthur Myers and James H. Steele, *Bovine Tuberculosis Control in Man and Animals* (St. Louis, Mo.: Warren H. Green, 1969): 57.
74. J. M. Grange, "Human Aspects of Mycobacteriumbovis," in *Mycobacteriumbovis*, ed. Charles O. Thoen and James H. Steele (Ames: Iowa State University Press, 1995): 29.
75. Grange, "Mycobacteriumbovis," 30. Speaking at the International Tuberculosis Congress in London in 1901, Koch argued that bovine TB merited little attention from public health authorities: "I should estimate the extent of infection by the milk and flesh of tubercular cattle and the butter made of their milk as hardly greater than that of hereditary transmission, and I therefore do not deem it advisable to take any measures against it." Quoted in Myers and Steele, *Bovine Tuberculosis*, 57.
76. Grange, "Mycobacteriumbovis," 31.
77. Bryder, *Below the Magic Mountain*, 133.
78. Myers and Steele, *Bovine Tuberculosis*, 60.
79. Smith, *Retreat of Tuberculosis*, 185.
80. Ibid., 186.
81. Myers and Steele, *Bovine Tuberculosis*, 75-84.
82. Although the downturn in TB mortality coincides with the influenza pandemic in the United States, Alfred Crosby offers little to suggest a causal relationship. Writes Crosby, "Spanish influenza was not a typical communicable disease in its choice of people to infect and kill. Unlike tuberculosis, typhoid fever, and venereal disease, it did not show a clear preference for the poor, the ill-fed, ill-housed, and shabbily clothed. Sometimes there was a discernible correlation between flu, pneumonic complications, and crowded living conditions... but by and large the rich died as readily as the poor." Immigrants, Crosby notes, had a higher death rate from

- influenza, however. Alfred W. Crosby, *America's Forgotten Pandemic: The Influenza of 1918* (Cambridge, England: Cambridge University Press, 1989): 227. Decreases in TB mortality following the federal bovine eradication program also occurred despite decreases in breast-feeding and increased use of infant formula. Rima D. Apple, "'To Be Used Only Under the Direction of a Physician': Commercial Infant Feeding and Medical Practice, 1870–1940," *Bulletin of the History of Medicine* 54 (1980): 402–417.
83. Pasteurization had become widespread in major American cities (those with populations of more than 75 000) by 1921. Small cities (with populations of less than 10 000) also pasteurized extensively by 1921. S. Henry Ayers, *The Present Status of the Pasteurization of Milk*, United States Department of Agriculture Bulletin No. 342, January 8, 1916; revised October 19, 1922, 5–7.
 84. Myers and Steele, *Bovine Tuberculosis*, xiv.
 85. *Ibid.*, xiv.
 86. George Rosen, *Preventive Medicine in the United States, 1900–1975, Trends and Interpretations* (New York: Prodist, 1977): 30.
 87. *Ibid.*, 30.
 88. *Ibid.*, 29.
 89. Rosen, *Public Health*, 336.
 90. Ayers, *Pasteurization of Milk*, 5–7.
 91. Myers and Steele, *Bovine Tuberculosis*, 70.
 92. George Jasper Wherrett, *The Miracle of the Empty Beds: A History of Tuberculosis in Canada* (Toronto: University of Toronto Press, 1977): 47.
 93. Thoen and Steele, *Mycobacterium bovis*, xx.
 94. Grange, "Mycobacterium bovis," 36, 38.
 95. Smith, *Retreat of Tuberculosis*, 175–194; Bryder, *Below the Magic Mountain*, 137–138, 246–247.
 96. Smith, *Retreat of Tuberculosis*, 175–194; Bryder, *Below the Magic Mountain*, 137–138, 246–247.
 97. Smith, *Retreat of Tuberculosis*, 186. From 1951 to 1965, Paris scientists estimated that approximately 3% of cases studied were caused by the bovine strain. Myers and Steele, *Bovine Tuberculosis*, 65.
 98. Smith, *Retreat of Tuberculosis*, 193.
 99. McKeown, *The Role of Medicine*, xii.
 100. Szreter, "Social Intervention," 33.
 101. McKeown, *The Role of Medicine*, xvi.
 102. McKeown, "A Sociological Approach," in *Medical History and Medical Care* (London: Oxford University Press, 1971): 4.
 103. Szreter, "Social Intervention," 33.
 104. Charles Webster, "The Origins of Social Medicine in Britain," *Society for the Social History of Medicine Bulletin* 38 (1986): 53.
 105. Szreter, "Social Intervention," 33.
 106. McKeown, *The Role of Medicine*, xiii.
 107. Eyler, *Newsholme*.
 108. *Ibid.*, 148–158.
 109. Hardy, *The Epidemic Streets*, 258, 262.
 110. *Ibid.*, 264.
 111. Newsholme, *The Last Thirty Years in Public Health*, 128; Hardy, *The Epidemic Streets*, 26.
 112. Prior to the bacteriological revolution of the 1870s and 1880s, public health in the United States was largely the domain of the sanitarians, reformers, and, occasionally, physicians devoted to eliminating the filth and squalor believed to cause disease via miasmas. Although social reformers and sanitarians maintained a presence in the public health movement after the bacteriological revolution, public health was increasingly dominated by a new set of public health professionals, many educated in northeastern medical schools and further trained in European laboratories. John Duffy, *The Sanitarians: A History of American Public Health* (Urbana: University of Illinois Press, 1990), and Paul Starr, *The Social Transformation of American Medicine: The Rise of a Sovereign Profession and the Making of a Vast Industry* (New York: Basic Books, 1982).
 113. The question of whether the "new public health" differed sharply from the old—long at the center of historical debate—hinges on different understandings of the relationship between public health and clinical medicine in the bacteriological era. One contingent of historians concludes that bacteriology helped to refine and focus the efforts of those 19th-century reformers who had developed a sense of public health as a communal concern. See Bruno Latour, *The Pasteurization of France* (Cambridge, Mass.: Harvard University Press, 1988). Duffy, *Sanitarians*, chap. 14; Rosen, *Public Health*, chaps. 7 and 8; Lloyd Stevenson, "Science Down the Drain: On the Hostility of Certain Sanitarians to Animal Experimentation, Bacteriology, and Immunology," *Bulletin of the History of Medicine* 29 (1955): 1–26; Barbara Gutmann Rosenkrantz, *Public Health and the State: Changing Views in Massachusetts, 1842–1936* (Cambridge, Mass.: Harvard University Press, 1972); Starr, *Social Transformation of American Medicine*; and Elizabeth Fee, *Disease and Discovery: A History of the Johns Hopkins School of Hygiene and Public Health, 1916–1939* (Baltimore: Johns Hopkins University Press, 1987).
- However, some scholars and social theorists, representing a second contingent, argue that bacteriology narrowed the scope of public health too much: the sanitarians had maintained a holistic concern about the individual in his or her environment that embraced health, moral status, and living conditions. In such analyses, the public health movement became closely associated with clinical medicine, which, in the 1960s and 1970s, was under sharp attack. Rosenkrantz, *Public Health and the State*, 179; Edward S. Golub, *The Limits of Medicine: How Science Shapes Our Hope for the Cure* (New York: Times Books, 1994); Daniel M. Fox, *Power and Illness: The Failure and Future of American Health Policy* (Berkeley: University of California Press, 1993); Rene Dubos, *Mirage of Health: Utopias, Progress and Biological Change* (New Brunswick, N.J.: Rutgers University Press, 1959, 1993); and Dubos and Dubos, *The White Plague*.
114. Elizabeth Fee, "Public Health, Past and Present: A Shared Social Vision," introduction to George Rosen, *A History of Public Health*, expanded edition (Baltimore: Johns Hopkins University Press, 1993): x.
 115. Susan Reverby and David Rosner, "Beyond 'the Great Doctors,'" in *Health Care in America, Essays in Social History*, ed. Reverby and Rosner (Philadelphia: Temple University Press), 1979: 4.
 116. David Musto, "Quarantine and the Problem of AIDS," in *AIDS: The Burdens of History*, ed. Elizabeth Fee and Daniel M. Fox (Berkeley: University of California Press, 1988): 67–85.
 117. Charles E. Rosenberg, "Disease and Social Order in America: Perceptions and Expectations," in *Explaining Epidemics and Other Studies in the History of Medicine* (Cambridge, England: Cambridge University Press, 1992): 259–260.
 118. *Ibid.*
 119. *Ibid.*, 260.
 120. Wilson, "Minnesota," 19–21.
 121. Nancy Tomes, "The Private Side of Public Health: Sanitary Science, Domestic Hygiene, and the Germ Theory," *Bulletin of the History of Medicine* 64 (1990): 509–539.
 122. See, for instance, McFarlane, "Hospitals, Housing, and Tuberculosis," 59.
 123. Newsholme, "Public Health Authorities," 456.
 124. Ronald Bayer et al. "Directly Observed Therapy and Treatment Completion for Tuberculosis in the United States: Is Universal Supervised Therapy Necessary?" *American Journal of Public Health* 88 (1998): 1052–1058.
 125. See note 37.